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Cerebral amyloid angiopathy-related infarcts imitating high-grade differentiation of a benign meningioma

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Published in:
BMJ Case Reports

DOI:
[10.1136/bcr-2015-211262](https://doi.org/10.1136/bcr-2015-211262)

Publication date:
2015

Document Version
Peer reviewed version

[Link to publication in Discovery Research Portal](#)

Citation for published version (APA):

Talarczyk, P., Kanodia, A. K., Ironside, J., & Mowle, D. (2015). Cerebral amyloid angiopathy-related infarcts imitating high-grade differentiation of a benign meningioma. *BMJ Case Reports*, 2015. <https://doi.org/10.1136/bcr-2015-211262>

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TITLE OF CASE
Cerebral amyloid angiopathy-related infarcts imitating high-grade differentiation of a benign meningioma.
DESCRIPTION <i>Up to 250 words summarising the importance of the image(s)</i>
<p>Meningiomas are divided by the World Health Organization (WHO) into three groups: benign (Grade I), atypical (Grade II) and malignant (Grade III). Although some overlapping features can occur, atypical meningiomas are more likely to exhibit heterogeneous density or intensity, heterogeneous contrast enhancement and relatively large perifocal oedema on CT and MR imaging.¹ Meningiomas typically receive their blood supply from dural arteries, although larger meningiomas can also have a blood supply from leptomeningeal or parenchymal branches. Cerebral amyloid angiopathy (CAA) results from β-amyloid deposition in the walls of leptomeningeal and cortical arteries.² It usually causes lobar intracerebral haemorrhage and often occurs in association with Alzheimer's disease. Rarely, CAA has been documented to be associated with inflammatory changes, sometimes thought to be co-existent primary CNS vasculitis or Giant cell arteritis, although most literature has focussed on probable inflammatory changes secondary to β-amyloid deposition, sometimes described as amyloid beta-related angiitis.³⁻⁶ We could not find any specific association of CAA with Wegener's granulomatosis in the literature.</p> <p>We present serial brain imaging findings describing the evolution of changes from a stable benign meningioma to a lesion imitating an atypical meningioma which was surgically removed and subsequently shown to be affected by amyloid angiopathy.</p> <p>A 59 year old female presented with a short history of right sided tinnitus, hearing loss and sinusitis. Her other significant known condition was Wegener's granulomatosis involving the lungs, diagnosed in another institution. MRI brain imaging in January 2011 (Figure 1) was negative for vestibular schwannoma, but demonstrated an incidental 14mm parafalcine meningioma in the left frontal region on the background of small vessel cerebrovascular disease. An MRI performed in March 2013 (Figure 2) due to acute neurological symptoms demonstrated extensive abnormalities in the right parieto-occipital lobe with further haemorrhage in left frontal lobe; these were assumed to be due to her known Wegener's granulomatosis, rather than from potential CAA. There was some increase in the size of the meningioma, which continued to show homogenous enhancement. A follow up MRI in April 2014 (Figure 3) showed further increase in size of the meningioma to 28mm, still with homogenous enhancement and without any significant surrounding brain changes.</p> <p>In November 2014 patient had further clinical deterioration with new onset right sided neurological symptoms. MRI (Figure 4) showed the meningioma as having a heterogeneous appearance with necrosis and peripheral enhancement. The adjoining brain also appeared grossly heterogeneous and necrotic with peripheral enhancement and significant mass effect. A Neurosurgical review suspected the changes to be suspicious of a high grade tumour progression and the meningioma was resected. Neuropathological examination found cerebral amyloid angiopathy involving vessels</p>

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in the meninges, cortex and within the meningioma, resulting in infarcts in a WHO grade I meningioma and adjacent cortex without any invasion or other features of atypical meningioma (Figure 5). No evidence of vasculitis or Wegener's granulomatosis was found. A follow-up MRI in February 2015 (Figure 6) demonstrated residual blood products with minimal post operative enhancement and resolution of most of the surrounding changes.

The current case shows unusual appearances in a grade I meningioma resulting from leptomeningeal, cortical and meningioma vessel involvement in amyloid angiopathy. These radiological appearances can easily be misinterpreted as an atypical meningioma. Involvement of blood vessels in a meningioma has not been previously described in CAA, which is otherwise a common condition in elderly and should be kept in mind if other associated brain changes, such as lobar haemorrhages, are present in patients with a meningioma.

LEARNING POINTS/TAKE HOME MESSAGE 2 to 3 bullet points – this is a required field

- 1) Infarcts in a WHO grade I meningioma can mimic high grade differentiation prompting neurosurgical concern.**
- 2) Cerebral amyloid angiopathy (CAA) can affect not only normal brain but also a pre-existing meningioma.**
- 3) Presence of brain changes consistent with CAA should prompt the suspicion of similar changes in a necrotic meningioma.**

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2014;44(1):86-92.

FIGURE/VIDEO CAPTIONS *figures should NOT be embedded in this document*

Figure 1: MRI January 2011. T2 axial (a) and Post contrast T1 axial (b) showing small left parafalcine meningioma (white arrow).

Figure 2: MRI March 2013. T2 axial (a), Pre and Post contrast T1 axial (b and c) showing some increase in size of parafalcine meningioma (white arrow). Further brain changes with haemorrhages are also present.

Figure 3: MRI April 2014. T2 axial (a) and Post contrast T1 axial and coronal (b and c) showing left parafalcine meningioma to have further slightly increased in size (white arrow).

Figure 4: MRI November 2014. T2 axials (a and b), pre contrast T1 axial (c) and post contrast coronal, sagittal and axial (d,e and f). Black arrow in a,c,d,e,f show the heterogeneously enhancing meningioma with necrosis. White arrow in a,b,d,e,f shows underlying brain abnormalities with heterogeneity, necrosis and oedema.

Figure 5: Immunohistochemistry for β -amyloid in the meningioma shows amyloid angiopathy in tumour vessel walls (brown), with splitting of the vessel wall and adjacent tumour infarction (lower left), x 100.

Figure 6: MRI February 2015 (post-op). T2 axials (a,b), FLAIR coronal (c), Post contrast T1 axials (d,e) and coronal (f) showing residual changes with some haemosiderin deposition and minor residual enhancement (white arrow).

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